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AURICULAR ORIGIN TO AURICULAR FIBRILLATION.

BY THOMAS LEWIS AND H. G. SCHLEITER.

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THE RELATION OF REGULAR TACHYCARDIAS OF AURICULAR ORIGIN TO AURICULAR FIBRILLATION.

BY THOMAS LEWIS* AND H. G. SCHLEITER.

(From the Cardiographic Department, University College Hospital Medical School).

IN the present communication we wish to record two cases of paroxysmal tachycardia which present features of exceptional interest. We utilize these two cases to illustrate the main thesis of this paper, namely, the close pathogenetic relation of regular tachycardia arising from an abnormal auricular focus and auricular fibrillation. The cases are of importance in that they support the general hypothesis that auricular fibrillation consists of an exaggeration of the phenomenon of pathological or heterogenetic impulse formation, which accounts for a large group of simple tachycardias arising in the auricle.

CASE 1. Paroxysms of auricular fibrillation. On one occasion a recorded paroxysm started as fibrillation and terminated as a regular tachycardia, springing from an ectopic auricular focus.

A. J., a cabinet maker of 28 years, was first seen on November the 11th, 1910, at the out-patient department at the City of London Hospital, in an attack of paroxysmal tachycardia. He has been under observation since that date and a number of paroxysms have been observed.

History. His mother died of "heart disease" 4 years ago; she is said to have had rheumatic fever. A letter from Dr. Hargrave, who attended her in her last illness, tells us that she succumbed to progressive failure of the heart, which resulted from asthma and chronic bronchitis. His brothers and sisters, of whom four are alive, have never suffered from tonsillitis, rheumatism, chorea or heart disease. His two children are alive and healthy. One sister died of enteric.

Personally he has had no previous illness, excepting measles as a child and an occasional cold. Rheumatic affections and syphilis are denied. He has always been moderate in the consumption of alcohol and tobacco.

Two years ago he had his first heart attack, and since that time has been subject to them at intervals of 1-3 months. He attributes the first attack to strain, as it came on while he was lifting a heavy piece of furniture. The attacks usually last 15-18 hours. Lately they have been more frequent and some have lasted longer. The shortest attack has been 7 hours, the longest 3½ days. He states that the attacks come on at any time; they have often commenced in the night; exertion predisposes to them. They are not influenced by diet, so far as he knows. They commence quite abruptly and end in the same fashion; the cessation is always marked by considerable relief.

The first symptoms of the attack are faintness and sweating; a few minutes later he is conscious of palpitation, and feels cold and sick. Vomiting is said to occur ½-1 hour later, and about the same time he is aware of a fixed pain in the front and lower part of the chest. A

* Working under the tenure of a Beit Memorial Research Fellowship.

little later, the pain increases and is more prominent at the pit of the stomach and over the right ribs. It also radiates to the shoulders. He becomes very exhausted, thirsty and short of breath. Flatulence is a prominent symptom of the attacks and may precede them.

Between the attacks he is fairly well. He can walk in comfort, but easily gets giddy. If he is sitting and rises suddenly, the giddiness may cause unsteadiness and he supports himself, fearing a fall. He sometimes gets a little breathless after walking up one short flight of stairs. The appetite is good and he sleeps well; the bowels are opened regularly. He suffers from wind, and has had vomiting during the past few months. On several occasions, bright blood has been brought up.

During the last six months, he has done no work. He has lost ten pounds in weight in the past two years.

Observations between the attacks.—The patient is well built and nourished. He has a healthy, in fact somewhat excessive, colour. There is no trace of cyanosis. The lungs are normal; the nervous and alimentary systems present no objective signs of disease. The urine is normal.

The liver dulness extends from the upper border of the 6th rib to the costal margin, its edge is not palpable. The spleen is not felt. The thyroid and lymphatic glands are normal. The limits of cardiac dulness are normal, lying 0 and $3\frac{1}{2}$ inches to right and left of the mid-sternal line. At the apex there is a short systolic thrill and a harsh systolic murmur. Otherwise the heart sounds are normal. The arteries are somewhat thickened; the systolic blood pressure varies between 70 and 120 mm.Hg., (Riva Rocci). The heart beats regularly as a rule (Fig. 1 and 8). Its rate lies between 50 and 78 per minute. At times, sinus irregularity has been present. The *a-c* interval is usually one-fifth of a second in length (Fig. 1). During the stays in hospitals, his temperature has always been normal.

Observations during the attacks.—On three occasions, he has been admitted to the City of London Hospital in attacks (November the 9th, 1910, December the 8th, 1910, and August the 4th, 1911); he stayed at Mount Vernon Hospital from February the 2nd till March the 11th, 1911, and on two occasions he has been taken in at University College Hospital for attacks (May the 31st and July the 1st, 1911). During his last stay at University College he developed an additional attack. On no other occasion has a paroxysm commenced during his hospital stays, most of which have been of several weeks or months duration.

The attacks which have been observed have all been of over 12 hours duration and on one occasion a paroxysm lasted $3\frac{1}{2}$ days.

Seen within half an hour of the onset he is already distressed. The face is pale and sunken; he is restless and complains of distress of breathing and precordial pain. As the attack progresses his symptoms become aggravated, some cyanosis appears, the respirations increase in rate and the pallor becomes more conspicuous. The heart dilates and the liver becomes swollen and pulsates. Dropsy has not been noticed, and there have been no physical signs of œdema of the lungs. He is salivated during the attacks, develops a cough and when they have lasted many hours commences to expectorate a little frothy mucus.

The swelling of the heart may be exemplified by the following observations. When originally seen during a paroxysm in November, 1910, the measurements on percussion were $\frac{1}{2}$ and $5\frac{1}{2}$ inches respectively. At the end of a long paroxysm of 24 hours duration, on May the 31st, 1911, they were 2 and $5\frac{1}{2}$ inches. 5 hours after the cessation of the same paroxysm they were $\frac{3}{4}$ and $3\frac{1}{2}$. On July the 1st, they were 2 and $6\frac{1}{2}$ inches, 2 hours after the onset. 7 hours subsequent to the offset of the attack they were 0 and 4. 15 minutes after the onset of the attack of July the 15th, they were 0 and $5\frac{1}{2}$, the patient lying quietly in bed; a few minutes' restiveness was accompanied by a decided increase in the dull area. A definite and striking change to $\frac{3}{4}$ and $6\frac{1}{2}$ inches was observed and it persisted. 1 hour later, the paroxysm continuing, the measurements were recorded at $1\frac{1}{2}$ and $6\frac{1}{2}$. Next morning the paroxysm had ended and the limits were 0 and $4\frac{1}{4}$.

The nature of the paroxysms.—The majority of the paroxysms have consisted of attacks of auricular fibrillation, during which the heart rate has varied between 155 and 200 per minute. At such times the venous pulse has been of the ventricular form. Paroxysms of this nature have been observed and recorded on five occasions; examples of the curves are shown in Fig. 3, 4 and 10.

The second form of paroxysm, which was recorded electrocardiographically on one occasion only, consisted of a regular tachycardia, the average rate of which was 140 per minute. It arose from an ectopic focus in the auricle and formed the termination of a long paroxysm which commenced as fibrillation (Fig. 2 and 9).

The graphic records of observed attacks are summarised in the following table.

1st paroxysm.	9/xi/1910.	Auricular fibrillation (lasting many hours). (Venous curves).
2nd paroxysm.	8/xii/1910.	Regular paroxysm. (Lasting approximately 12 hours). (Venous curves taken but mislaid).
3rd paroxysm.	25/ii/1911.	Auricular fibrillation. (Lasting 10 hours).
4th paroxysm.	31/v/1911.	Auricular fibrillation. (Lasting 12 hours). (Electrocardiograms, 3 leads).
5th paroxysm.	1/vii/1911.	Auricular fibrillation. (Lasting $3\frac{1}{2}$ days). (Electrocardiograms, 3 leads, Fig. 10.)
	2/vii/1911.	Tachycardia of auricular origin. (Electrocardiograms, 3 leads, Fig. 9 and venous curve, Fig. 2).
6th paroxysm.	15/vii/1911.	Auricular fibrillation. (Lasting about 10 hours). (Polygraphic curves and electrocardiograms, 3 leads).
7th paroxysm.	4/viii/1911.	Auricular fibrillation. (Lasting approximately 24 hours). (Polygraphic curves)
Control curves of the normal rhythm were taken on a number of occasions, (1/vi/1911, 4/vii/1911, 14/vii/1911, 19/vii/1911, 21/vii/1911, 24/vii/1911, etc.).		

The polygraphic curves are exemplified by Fig. 1-4. The curves are not treated chronologically. Fig. 1 shows venous and radial curves, taken on July the 21st, 1911, during a period when the pulse was slow (63 per minute) and regular. The *a-c* interval is one-fifth of a second.

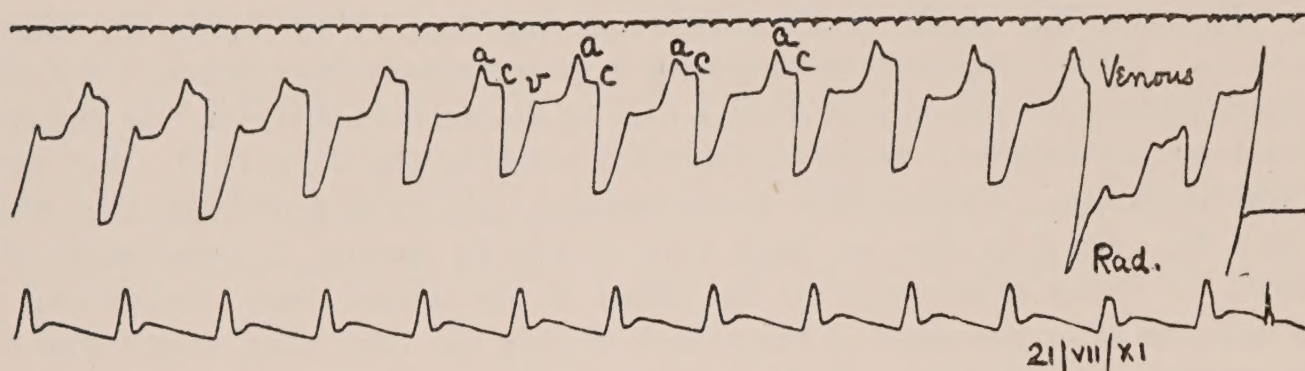


Fig. 1. *CASE 1.* A polygraphic curve taken while the heart's action was slow and regular. The *a-c* interval is $1/5$ sec.. The pulse rate is 63 per minute. (July the 21st, 1911). The time-marker of all the polygraphic curves is in $1/5$ sec..

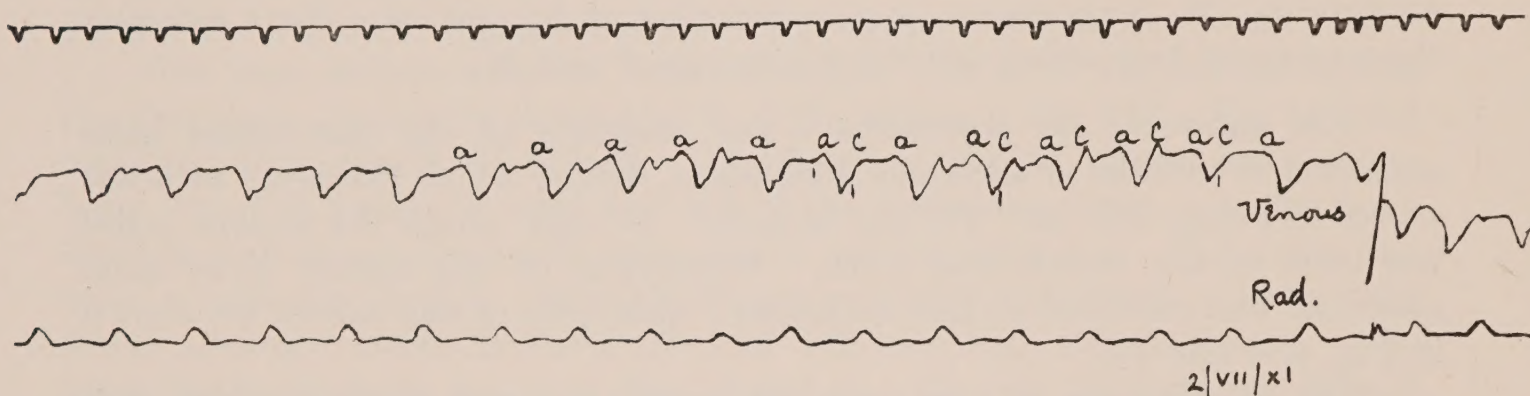


Fig. 2. *CASE 1.* A polygraphic curve taken during the long paroxysm observed on July the 2nd, 1911. The heart's action is regular; the rate is 142 per minute; the *a-c* interval is a full $1/5$ sec..

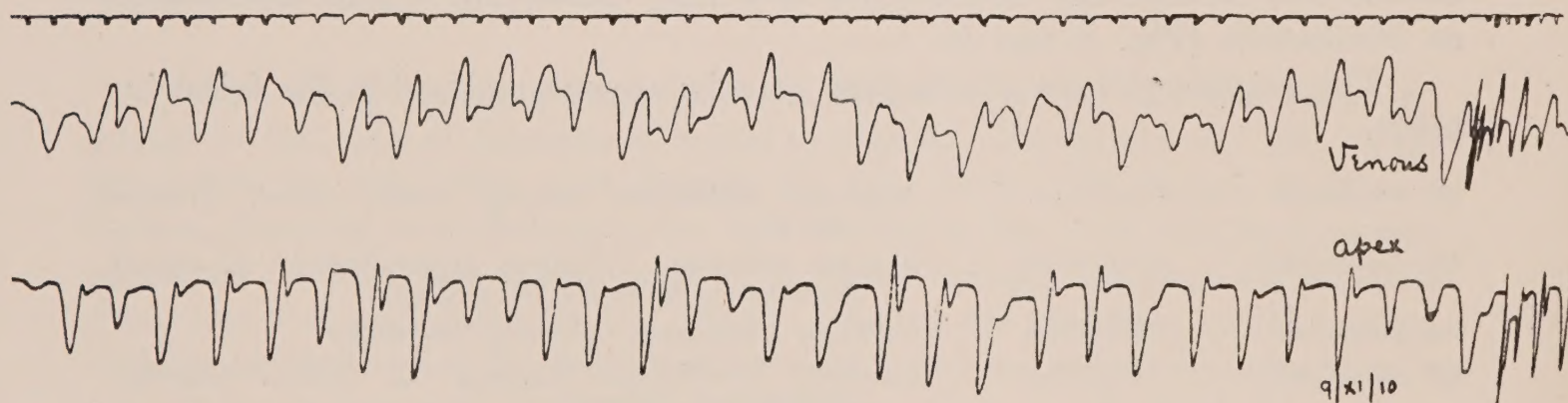


Fig. 3. *CASE 1.* A jugular and apex curve, taken during a paroxysm of tachycardia, (November the 9th, 1910). The heart's action is irregular; the rate is 165 per minute. The jugular pulse is of the ventricular form.

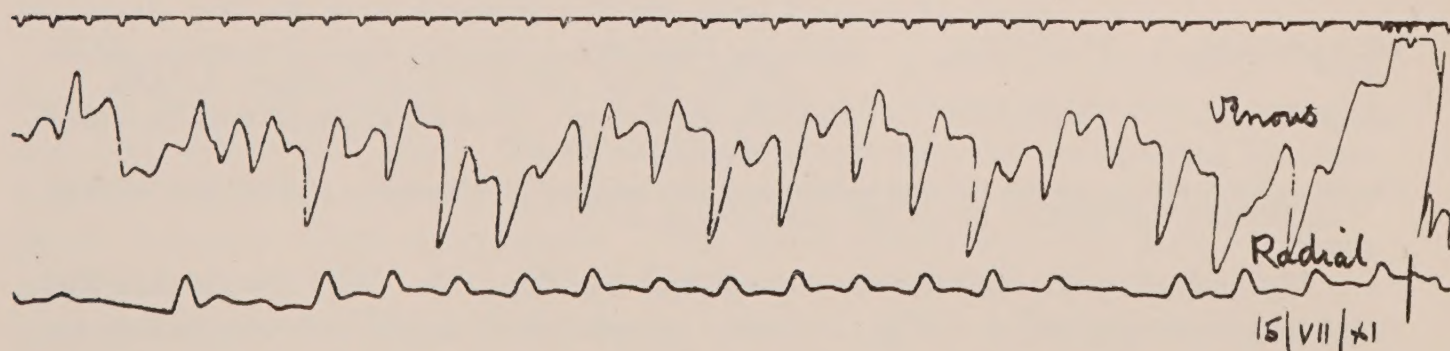


Fig. 4. *CASE 1.* A jugular and radial curve taken during a paroxysm of tachycardia, (July the 15th, 1911). The heart's action is irregular; the rate is 155 per minute. The jugular pulse is of the ventricular form.

Fig. 2 shows venous and radial curves taken at the end of a paroxysm on July the 2nd, 1911. The heart's action is regular; its rate is 142 per minute. The *a* wave is small and obscure. The *a-c* interval is one-fifth of a second. A number of observations were made upon the effect of certain events upon the rate of this paroxysm. Repeated swallowing had no influence on the rate. Changing from the sitting to the lying posture did not influence the heart's rate by more than two beats per minute. Repeated suspension of respiration had little or no influence upon the rate.

Fig. 3 and 4 are examples of curves taken during paroxysms on November the 9th, 1910 and July the 15th, 1911, respectively. Fig. 3 shows a venous and apical curve, Fig. 4 shows a venous and radial curve; in both, the heart is beating irregularly and the venous curve is of the ventricular form.

Electrocardiograms of the three separate mechanisms are shown in Fig. 8, 9 and 10. Each figure includes a series of three leads, marked *I*, *II* and *III* respectively. These represent Einthoven's usual leads in each instance: *I*, right arm to left arm; *II*, right arm to left leg; *III*, left arm to left leg. In each series the second lead is standardized so that 1/1,000 volt equals 0.6 cm. in the reduced curve.

The curves are not treated in chronological order. They are simply given as examples of the mechanisms which have been observed.

The normal mechanism has been recorded electrocardiographically on two occasions, on June the 1st, 1911 and on July 4th, 1911. The curves are identical for the two dates. Speaking of Fig. 8, each cardiac cycle of the regular mechanism shows auricular (*P*) and ventricular (*R*, *S* and *T*) representatives. *P* is prominent and bifurcates at its summit in leads *I* and *III*. The *P-R* interval is 0.14 sec.. The rate of the heart beat is 65 per minute.

This series should be compared with the two following series, Fig. 9 and 10. Fig. 9 was taken during the regular paroxysm of July the 2nd, 1911. Lead *I* shows *R* and *T* summits. *T* is inverted. (The first lead of the normal mechanism, Fig. 8, shows no *T* summit). *P* is very indistinct in this lead. In the second lead *R* and *T* waves are well represented; *R* is of less amplitude than in the normal mechanism. In Fig. 9 *II*, *P* is clearly represented as a complex curve, consisting of downward, upward and downward deviations. The third lead shows a diminished *R*, an increased *S* and an upright *T*. *P* is very similar to that found in lead *II*. The rate of heart-beat in this figure is 140 per minute. The *P-R* interval is approximately 0.18 sec. in duration. Comparing the two series of curves, it is seen that the ventricular complexes of the tachycardia and normal rhythm, Fig. 8 and 9, correspond fairly closely in the respective leads; the variations are chiefly in the amplitude of the various summits, and in an occasional inversion of *T*. The resemblance is sufficiently close to allow us to conclude that the origin of the heart beat is supraventricular during the tachycardia. The auricular origin of the paroxysm is proved by the presence of the clear auricular representatives in leads *II* and *III*. Nevertheless, the paroxysm

has originated in a focus at some distance from the natural pace-maker, as is indicated by the anomalous appearance of these auricular representatives. At the time when the electrocardiograms of the regular paroxysms were obtained, the movements of the string were watched and occasional premature contractions, interrupting the paroxysm itself, were observed, but were not recorded. The ventricular portion of such cycles seemed to give exactly similar electric complexes to those of the remaining paroxysmal cycles. The premature contractions, therefore, were presumably of auricular origin also.

The third mechanism is shown in Fig. 10, curves taken on July the 1st, 1911. They are from the beginning of the paroxysm of which Fig. 9 shows the termination. Similar curves were obtained on May the 31st, 1911, and on July the 15th, 1911. The ventricular complexes in Fig. 10 are similar in the three leads to those of the corresponding leads in Fig. 9. The beats are placed at quite irregular intervals throughout the curves. The rate is variable, but at times it reaches nearly 200 per minute. *P* summits are entirely absent in all the curves. No two adjacent cycles are exactly alike. The slight variation from cycle to cycle is due to the presence of the characteristic oscillations of auricular fibrillation, which are most conspicuous where the diastoles are relatively long, (*f.f.*)

CASE 2. Short and long paroxysms of tachycardia; the majority of the paroxysms were short and consisted of regular tachycardias springing from an abnormal focus in the auricle. They were occasionally interrupted by beats from a second abnormal auricular focus. On one occasion, curves showing the passage from the regular mechanism to auricular fibrillation were obtained.

G. T., a married man, aged 71, came to the City of London Hospital on May the 28th, 1911, complaining of chronic cough, pain in the chest and lower part of the back, together with occasional stiffness in the right upper limb.

History. The father and mother died at the ages of 81 and 77 respectively. Five brothers are dead, one, it is said, of heart disease; one sister is alive and in good health. He has had twelve children, four of whom died in infancy; of the remaining eight, one is tuberculous and the others are healthy. The wife, aged 52, has had one miscarriage. The patient, formerly a wood carver, is now an old age pensioner. In infancy he had measles and whooping cough. At the age of 51, he was in the Bethnal Green Infirmary for pleurisy and inflammation of the lungs. A few years later he had rheumatic fever. He states that he was laid up three years ago with a second attack of pneumonia. On the whole he affirms that he has enjoyed fair health. He denies venereal infection. In his younger days he drank ale freely. As a smoker, he has been moderate.

His present illness dates from 1908, when he first experienced cough and pain in the chest and lower part of the back. For the most part, pain has been referred to the precordium. It is gnawing in character and is related to meals. Sometimes it is said to have been so severe as to make breathing difficult, a feature which suggests for it a cardiac origin. Shortness of breath and palpitation of the heart have been noticed upon exertion. He has suffered from, and still has, a chronic cough with a slight expectoration of mucus. There has been no hæmoptysis. His appetite is poor, he sleeps badly and gets up at night to pass water.

Observations between the attacks of tachycardia.—The patient is an undersized and poorly nourished individual. His skin is sallow. There is no oedema or cyanosis. The veins of the lower limbs are varicose. There is extensive pyorrhœa. The thyroid is not enlarged. There is evidence of a mild degree of emphysema. There are fine crepitations at both bases. The nervous and alimentary systems present no signs of importance.

The heart's apex beat is in the 5th interspace, $4\frac{3}{4}$ inches to the left of the middle line. There is systolic retraction in the 5th interspace internal to the apex. The limits of the heart's dulness lie 0 and $4\frac{3}{4}$ inches to the right and left of the mid-sternal line, respectively; they do not change with posture. The heart's sounds at the apex are clear and show no alterations. There is a musical systolic murmur after the first sound, and it is transmitted to the left axilla. The aortic second sound is somewhat increased in intensity. A regular heart rhythm is interrupted by premature beats, which are fairly frequent; most of them reach the wrist. The arteries are thickened but not tortuous. The pulse is of good volume. The systolic blood pressure is 120 mm. Hg..

The upper level of liver dulness commences at the 7th rib; the lower limit is at the rib margin. Neither liver nor spleen is to be felt. There is no abdominal tenderness or other abnormality. The urine is normal. A Wassermann reaction is negative.

Observations upon the paroxysms.—Short paroxysms of tachycardia have been recorded upon almost all of the numerous occasions of examination. Nevertheless they are relatively infrequent; as a rule two or three paroxysms occur during the two or three hours of investigation. They last for a time varying from two or more seconds to an hour. The majority of the paroxysms last from 10 to 20 seconds. The patient is absolutely unconscious of onset and offset and is unaware of the paroxysm while it is present. Apart from posture, we have been unable to ascertain that any factor influences the presence or absence of paroxysms to any real extent; they appear to be more frequent in the standing and sitting postures.

With a solitary exception, an exception which will be described in detail, the recorded paroxysms have consisted of regular tachycardias, the rate rising from the normal of 80 or 90 beats per minute to 122 or 170 per minute. The usual paroxysmal rate is between 135 and 145 per minute. These paroxysms are due to new rhythms arising from an abnormal point in the auricle. On one occasion, after a prolonged period of regular tachycardia, the mechanism altered to fibrillation. The preceding regular tachycardia was the most rapid observed, namely 170 beats per minute. The rate during the fibrillation was variable, it rose at times to 190 beats per minute.

Description of the graphic records.—Examples of paroxysms as they appear in radial pulse curves are shown in Fig. 5 and 6. Fig. 6 shows a

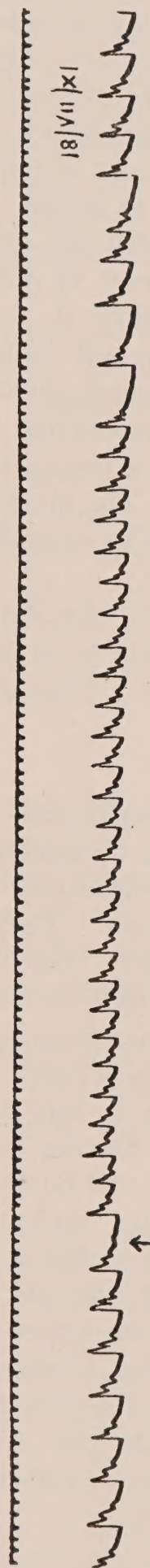


Fig. 5. *CASE 2.* A paroxysm of regular tachycardia taken on July the 18th, 1911. It lasts 14 seconds. The first beat of the paroxysm fails to affect the pulse. The paroxysm is followed by a long post-paroxysmal pause and a period of retarded pulse rate, which is interrupted by a single premature beat. The time-marker in all sphygmographic curves is in 1/5 sec..

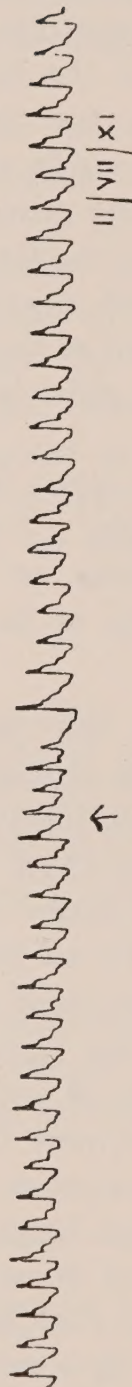


Fig. 6. *CASE 2.* A short paroxysm of 4 beats taken on July the 11th, 1911. The paroxysm starts abruptly and finishes equally suddenly. It is followed by a post-paroxysmal pause and by a short period of pulse retardation.

paroxysm of 4 beats only. Fig. 5 is an example of a paroxysm of longer duration. Most of the paroxysms have been similar to that portrayed in this curve. The onset and offset are quite abrupt. The offset is marked by a considerable degree of pulse slowing which vanishes within a few cycles; this phenomenon has been observed in many previous cases. A single premature contraction interrupts the normal rhythm shortly after it is resumed (in Fig. 5).

The premature contractions which interrupt the slow rhythm are of four kinds. Very occasionally the points of origin lie in the ventricle. The electrocardiograms have shown that some of these premature ventricular beats arise in the left or apical portions of the ventricle, some in the right or basal portions of the ventricle; we do not consider it necessary to reproduce the figures. The majority of the premature beats have arisen in the auricle; and they originate in two auricular foci, both of which lie at a distance from the pacemaker. The two types are illustrated by Fig. 12 and 15. In Fig. 12 the normal rhythm, to which the first three and the last cycle belong, is interrupted by a single premature contraction, arising in the auricle and represented by *P*, *R* and *T* summits. The *R* and *T* summits of the premature beats are similar to those of the rhythmic beats. The *P* summit of the premature beat is partly iso-electric, but is mainly a deviation in the upward direction; it differs from the *P* summits of the rhythmic beats. The second type of premature auricular contraction is shown in Fig. 15. As in the first type, the ventricular complex is similar to that of the rhythmic beats. On the other hand, the auricular representative is of different form. It consists of a deviation in the downward direction.

The short and regular paroxysms, a very large number of which have been observed, have been of one type. They are illustrated by Fig. 13, 14 and 16. They consist of a succession of beats arising in the auricle at the same point from which the second type of single premature beat is shown to arise (Fig. 15). The centre of such a paroxysm is published in Fig. 13, in which the rate is approximately 140 per minute. The cycles consist of a downwardly directed auricular representative *P*, and the usual summits *R* and *T*, corresponding to the ventricle and of similar form to those of the rhythmic beats. The termination of a similar paroxysm is shown in Fig. 14. In a very large number of the observed paroxysms, single beats springing from the auricle, but arising in a focus which is distinct from that at which the usual paroxysmal beat originates and from that at which the rhythmic beat of the slow periods arises, interrupt the paroxysm itself. Two examples of this phenomenon are shown, namely, the last cycle but one in Fig. 13 and the last cycle of the paroxysm in Fig. 14. The majority of these interrupting beats are slightly premature in relationship to the rhythm of the paroxysm. This is shown in Fig. 14, but in Fig. 13, the interrupting beat falls at its proper time. A comparison of the interrupting beats with the premature beat of Fig. 12 shows that they arise from the same focus. It has been remarked that they frequently terminate a paroxysm.

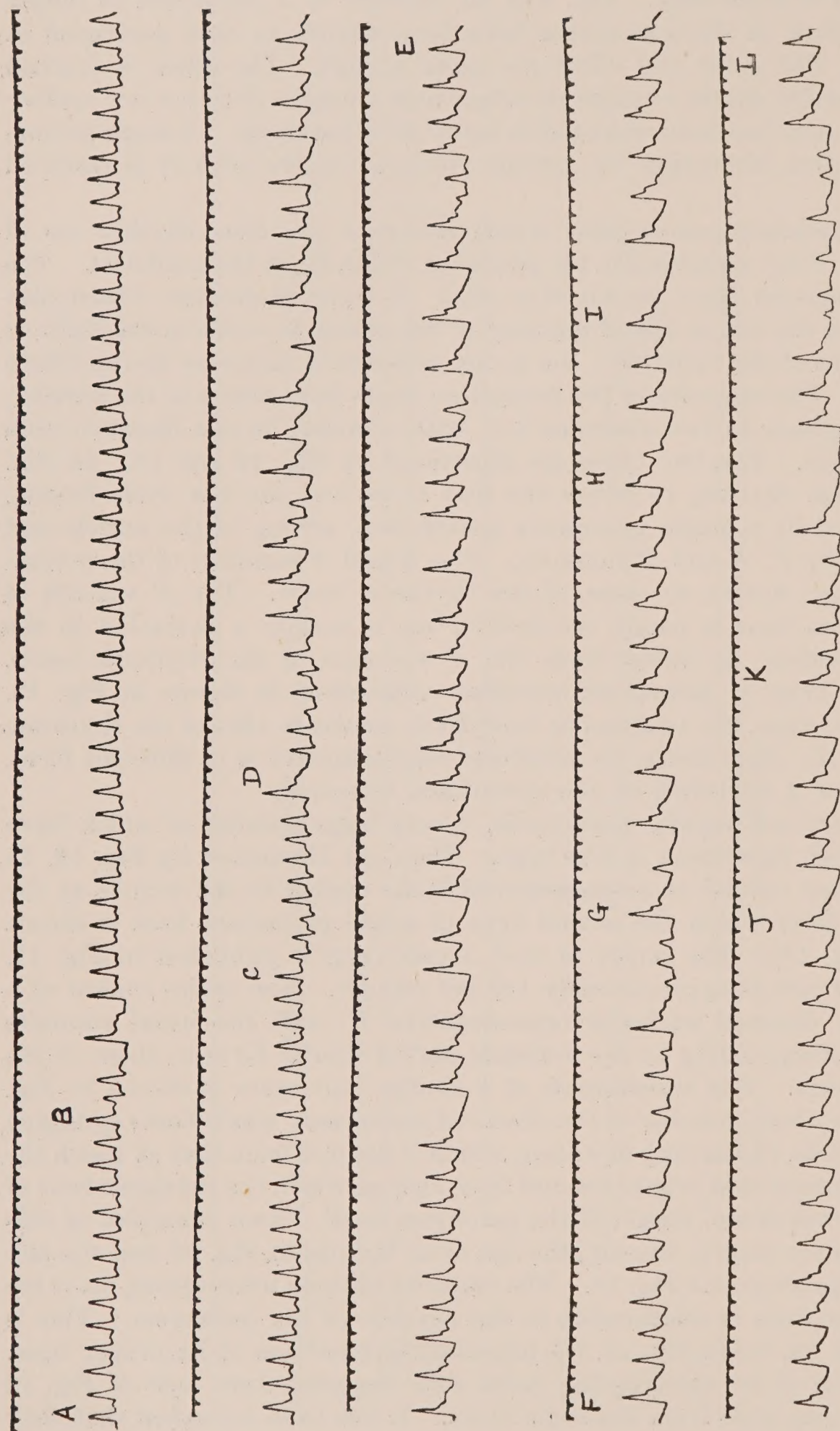


Fig. 7. CASE 2. Portions of a continuous sphygmographic curve taken from the radial artery on July the 18th, 1911. A regular paroxysm preceded it up to the point marked *A*; it is continued, with the exception of a short irregular period at *B*, up to *C*. At this point the pulse becomes irregular and at *D* the irregularity is complete. At *D* the auricle is fibrillating; the fibrillation is continued between *D* and *E*. Between *E* and *F* a portion of similar curve of 45 seconds duration has been excised. The fibrillation terminates at *G*, where the normal rhythm, interrupted by solitary premature beats, takes its place. Premature beats are seen at *H*, *I* and *J*. At *K* fibrillation sets in once more and the commencement of an attack of over one hours' duration is shown.

To sum up, the paroxysms consist of beats which arise in a single abnormal focus in the auricle and these abnormal or ectopic rhythms are disturbed by interruptions from a second ectopic focus. It is noteworthy that both the types of beat found during the paroxysms are also found as interruptions of the normal slow rhythm. Briefly, the patient possesses two foci of irritation in the auricle, each of which lies at a distance from the pacemaker. Both foci generate single new impulses and one of them generates impulses in succession.

We now pass to a description of certain events recorded on the 18th of July, 1911, and we may conveniently commence with a description of a continuous sphygmographic curve taken over a period of several hours on the afternoon of that day, (Fig. 7). When the patient first came under observation, the pulse was very irregular, but by the time the sphygmograph had been arranged, it was regular and beating at an approximate rate of 170 to 180 per minute. The patient exhibited a regular paroxysm of the usual form, though of somewhat faster rate. The regular paroxysm ran for 72 seconds, up to the point marked *A* in Fig. 7. At the point marked *B*, it became irregular for a second or more. The continuous curve is given in successive strips. At *C* the pulse shows some irregularity. At *D* it becomes very irregular and assumes the form which is so characteristic of auricular fibrillation. The same mechanism is continued from *D* to *E*; between *E* and *F* a portion of the curve of 45 seconds' duration and of precisely similar form has been excised. From *F* to *G* the fibrillation continues. At *G* the normal rhythm is resumed, but is interrupted by occasional premature beats at *H* and *I*. The normal rhythm is continued up to the point marked *J*. Occasional premature contractions then appear, and at *K* the complete irregularity, which is characteristic of a fibrillating auricle, commences again and continues to the end of the tracing (*L*). Following upon this strip, the completely irregular pulse continued for 69 minutes. The normal rhythm reappeared for 32 minutes. It was then interrupted by a paroxysm lasting 14 seconds. This paroxysm is shown in Fig. 5. A period of normal rhythm of 6 minutes' duration followed, and this in its turn was succeeded by a regular paroxysm of 21 seconds duration. The normal rhythm which followed was watched for 25 minutes, when the observations came to an end.

During the progress of the continuous radial curve, a large number of electrocardiograms were obtained. The first curve (Fig. 16) was taken somewhere between the points *A* and *D* in Fig. 7. The paroxysm consists of a succession of beats of a similar character to those shown in Fig. 9. The two curves differ mainly in rate. A second electrocardiogram (Fig. 17) was taken shortly after the onset of the complete irregularity (Fig. 7 *D*). Fig. 18 was taken during the stage of complete irregularity following *L* (Fig. 7).

The electrocardiograms, Fig. 17 and 18, testify conclusively to the presence of fibrillation of the auricles. The ventricular beats, represented by *R* and *T* summits of the usual form, are scattered throughout the curves

at irregular intervals. The summits *R* are separated by portions of curve which differ slightly from each other from cycle to cycle; this variation is due to the presence of the characteristic oscillations of auricular fibrillation, which are most marked in Fig. 18 (*f, f*).

In most instances of paroxysmal tachycardia arising in the auricle, whether the paroxysms originate in a single focus and are regular or whether the paroxysms originate in fibrillation and are irregular, it is noted that the height of the peak *R* during the paroxysmal stage is increased as compared with the normal. In the present case we have been unable definitely to ascertain the presence of this phenomenon, and probably this has been due in some measure to a phenomenon of an exceptional kind. In a number of radial curves obtained from the patient, very distinct evidence of a *pulsus alternans* has been obtained from time to time; and in electrocardiographic curves taken during the normal periods, a considerable variation in the amplitude of *R* has been frequently observed; this variation has not necessarily occurred in alternate fashion. This is well shown in Fig. 11, in which two *R* summits of large amplitude are followed by two *R* summits of lesser amplitude. Similar variations of amplitude have been frequently met with during the paroxysms. A notable example is shown in Fig. 13. A comparison of amplitudes during normal and paroxysmal periods has consequently been difficult or impossible.

DISCUSSION.

As we stated in our introductory remarks, the main object of this paper is to correlate two conditions, regular paroxysms of tachycardia arising in the auricle and auricular fibrillation. But while this is the main proposition before us, it is necessary to go somewhat further afield, and to collect certain accessory evidence bearing upon the production of pathological beats in the auricle. The view that tachycardia of auricular origin and auricular fibrillation have a common pathogenetic basis is but part of a more general conclusion. This general conclusion was dealt with by one of us at considerable length in a recent publication.¹⁰ The view is held in respect of both divisions of the heart, namely, auricle and ventricle; it is that there are three chief stages in the production of those pathological impulses which have been termed heterogenetic. There is the single and isolated heterogenetic impulse which gives rise to the simple premature contraction; there is the series of heterogenetic impulses giving rise to a new regular and fast rhythm; and, finally, there is the condition in which multiple foci are active and in which the musculature is so disturbed by impulses thrown out from multiple centres, that co-ordinate contraction is impossible and a state known as fibrillation ensues. The conception is that the three stages in the disorder of a division of the heart are due to a single underlying phenomenon, namely heterogenetic impulse formation. Variation in the degree of disorder is attributed to a variation in the degree of muscular

irritability in response to the exciting agent, or to a variation in the strength or distribution of the exciting agent. While the actual observations of this paper are more especially directed towards the proof of the close inter-relationship of new rhythms and fibrillation, it may be well to bring forward, in a general survey, the main facts upon which the general conclusion rests and to treat the special proposition of this communication as an integral part of it. The evidence is summed up in the following paragraphs.

1. When a weak faradic current is applied to the auricle or ventricle, and this current is gradually strengthened, a series of events is recorded. Single premature beats are first seen. An increase of current produces a regular tachycardia. Finally, with the strongest current, the muscle passes into fibrillation.

2. Many interferences with the muscle of the heart, as for example the administration of light percentages of chloroform or the injection of adrenalin⁴ or obstruction of the coronary vessels,⁶ awaken similar series of events. Single interruptions of the normal rhythm appear and these are succeeded by tachycardia and fibrillation, originating in the muscle affected.

3. A recent observation connects the tachycardias and fibrillation. It is well known that if an auricle be subjected to a fairly powerful faradic current and fibrillation is induced, it frequently happens that, at the cessation of stimulation, the fibrillation continues for some little while. The phenomenon is referred to by German writers by the apt term "Nachflimmern." We have observed the repeated replacement of this "Nachflimmern" by a regular tachycardia.

4. If a regular tachycardia is produced in the auricle by means of a weak faradic current, vagal stimulation will sometimes convert the tachycardia into auricular fibrillation.¹⁰

We may now sum up certain clinical evidence.

5. It is a usual experience in observations upon clinical cases of tachycardia of auricular or supraventricular* origin to find that the slow periods which separate the paroxysms are interrupted by premature beats of a precisely similar† nature. Our own observations include six cases, of which five have been previously recorded, ^{5, 7, 8 and 9} and the second case of the present communication forms the sixth. Similar observations have been made by Cohn¹ and Laslett.³

6. Curves showing intermediate or transitional conditions between single premature contractions and long paroxysms of tachycardia are not

* We insert the word supraventricular, because in some of the cases it has been impossible definitely to establish the auricular origin of the paroxysm, although their generation in this division of the heart is probable in most of the instances referred to.

† Precisely similar in so far as the graphic methods employed are concerned.

infrequent in given cases. An example is shown in Fig. 5, where four premature beats follow each other in succession. Other examples might be referred to (Lewis,⁹ Cohn¹ and Laslett's³ cases).

7. In patients who exhibit paroxysms of auricular fibrillation, the slow rhythm, which separates such paroxysms, is frequently interrupted by single premature auricular contractions. Two instances of this nature have come under the observation of one of us and have already been recorded.⁸ A series of similar cases was previously reported by Mackenzie.¹¹ An important instance of an allied type of case has been described by Hewlett,² a case in which certain transitions between the one form of irregularity and the other were observed.

8. Paroxysms of regular tachycardia of auricular or supraventricular origin and paroxysms of auricular fibrillation may occur in one and the same case. The writers have had an opportunity of studying a series of such cases and the two cases recorded in this paper are examples of this association. Moreover, the direct passage of regular paroxysms into fibrillation or fibrillation into regular paroxysms has been repeatedly observed. The original observation was made upon a case in which short paroxysms of regular tachycardia arising in the auricle interrupted the normal rhythm.⁸ On one occasion while electrocardiographic curves were being taken, the mechanism changed from tachycardia of auricular origin to auricular fibrillation, and subsequently the regular paroxysm was resumed. The same phenomenon probably occurred in Hewlett's case² (cp. Fig. 5 of his paper). It has also been recorded in a case described by Mackenzie¹² and in a case described by Turnbull;¹³ (in the latter the actual passage from one to the other was not caught). In the former the passage from one to the other was frequently caught. In the present paper, we have added two cases: in *CASE 1* a long paroxysm of many hours duration commenced as auricular fibrillation and terminated as a tachycardia of auricular origin. In *CASE 2*, a patient subject to numerous tachycardial paroxysms arising in the auricle, the passage of a paroxysm of this form into auricular fibrillation was observed and is shown in Fig. 7.*

9. The frequent association of auricular fibrillation with mitral stenosis and rheumatic heart disease is now a well recognised fact. In our own series, which comprises twelve cases of paroxysmal tachycardia of auricular or supraventricular origin, five were cases of mitral stenosis and in one of the remaining seven cases there was a history of rheumatic fever. We cite these figures for the purpose of showing that regular tachycardias and fibrillation are met with in a common class of cases.

While we divide heterogenetic impulse formation into three grades, the isolated premature beat, regular tachycardia and auricular fibrillation,

* Two new instances of the passage of regular paroxysms of tachycardia of auricular origin into auricular fibrillation have been observed by one of us, since these pages were written.

the division is an arbitrary one, and transitions from one stage to another occur and have been already referred to. Hewlett's case is a notable instance. *CASES 1* and *2* of the present communication may also be cited in this connection. In *CASE 1*, the regular paroxysm was interrupted by premature beats, which presumably were also of auricular origin, although they were not recorded electrocardiographically. *CASE 2* provides a clear instance of the transition. Paroxysms of regular tachycardia, generated in an ectopic focus, are interrupted by single beats, most of them premature in relationship to the paroxysm itself, springing from a separate ectopic focus. The presence of two active extraneous foci of impulse formation in the auricle, and the ultimate passage of a paroxysm into fibrillation in this patient, has a peculiarly significant bearing upon the question of the production of auricular fibrillation from multiple auricular foci. Whether the short period of irregularity recorded in Fig. 7 (at the point *B*) is a further transition or not we are unable to ascertain positively in the absence of the corresponding electrocardiogram, but we believe this to be the case.

We may sum up the previous discussion in the statement that there is a rapid accumulation of evidence, which shows the close association of the modes of genesis of three forms of disturbance of the normal cardiac rhythm, namely, premature contractions, paroxysms of regular tachycardia and fibrillation. The hypothesis represents the relation in more tangible form; the three forms of disturbance may be regarded as the expression of heterogenetic or pathological impulse formation of three grades, the production of single and isolated impulses, the occurrence of a series of impulses from a single focus and finally the activity of a number of such foci.

It is perfectly true that certain cases show one or other mechanism alone. It is also true that the connecting links in the individual case are often imperfect or absent; thus, cases of auricular fibrillation may apparently show intervening periods of slow and normal heart action which are perfectly regular. It is possible, nay probable, that records of the actual passage of normal mechanism to fibrillation in these last cases will ultimately reveal transitions, but we freely admit the possibility that such transitional curves may not occur, and that the changes may be abrupt from one to the other. An abrupt change from auricular fibrillation to a normal mechanism is indeed the rule in experiments upon healthy hearts, but we cannot see that these observations appreciably affect our general contention.

SUMMARY.

1. Two instances of paroxysmal tachycardia are recorded. In the first case, the paroxysms usually consisted of auricular fibrillation; on one occasion, at least, the passage of the fibrillation into a regular tachycardia

of ectopic auricular origin was observed. In the other instance, the paroxysms consisted of tachycardia of ectopic auricular origin; on one occasion the passage of such a tachycardia into fibrillation and the subsequent resumption of the normal rhythm, interrupted by regular paroxysms, was observed.

2. The regular tachycardias in these two cases were interrupted by premature contractions, which in one case probably, and in the other case certainly, came from a separate and ectopic auricular focus. The significance of this observation and its relation to the passing of regular tachycardia into fibrillation is discussed.

3. A detailed account of the evidence, which leads us to the view that the single premature contraction, ectopic tachycardia and fibrillation have a similar pathogenesis, is given.

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- ⁵ LEWIS. *Heart*, 1909, I, 43.
- ⁶ LEWIS. *Heart*, 1909, I, 98.
- ⁷ LEWIS. *Heart*, 1909, I, 262.
- ⁸ LEWIS. *Heart*, 1910, I, 306, (Case 10, 11 and 15, and Fig. 9).
- ⁹ LEWIS. *Heart*, 1910, II, 127.
- ¹⁰ LEWIS. "The Mechanism of the Heart Beat," etc., London, 1911. Chapters XI and XVI, and Fig. 145.
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- ¹² MACKENZIE. *Heart*, 1911, II, 273.
- ¹³ TURNBULL. *Heart*, 1911, III, 89.

Fig. 8, 9 and 10. *CASE I.* Three series of curves from a case of paroxysmal tachycardia, showing separate mechanisms. Each series comprises the three customary leads; right arm to left arm (*I*), right arm to left leg (*II*), and left arm to left leg (*III*). In each series lead *II* is standardised, so that 1/1000 volt = 0.6 centimetres in the reduced curve.

Fig. 8. Showing the mechanism of the heart on June the 1st, 1911, when the pulse was regular and when its rate was 65 per minute. The *P-R* interval is .14 sec.. The time-marker is in 1/30 sec.. (June the 1st, 1911).

Fig. 9. Showing the mechanism on July the 2nd at the end of the long paroxysm. The heart beats are regular, at the rate of approximately 140. The ventricular complexes are of the type induced by supraventricular impulses; each is preceded by an anomalous auricular complex *P*. The *P-R* interval is 0.18 sec.. The time-marker is in 1/5 sec.. (July the 2nd, 1911).

Fig. 10. Showing the mechanism on July the 1st, shortly after the onset of the long paroxysm. The beats are irregularly placed; the rate reaches 193 per minute. The ventricular complexes are all of the type attributable to supraventricular impulses. Co-ordinate contractions of the auricle are not represented. *P* is replaced by the oscillations (*f,f*) which characterise fibrillation of the auricle. The time-marker is in 1/5 sec.. (July the 1st, 1911)

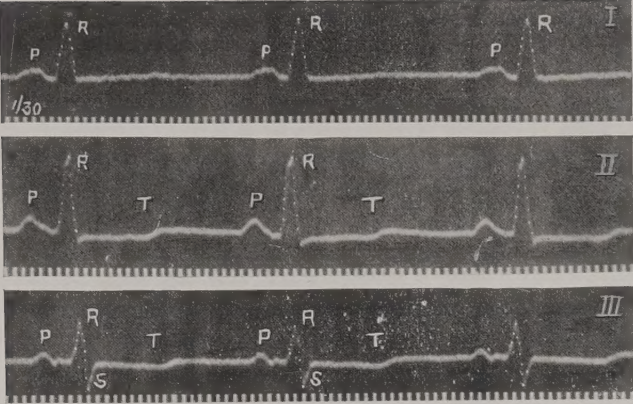


FIG. 8.

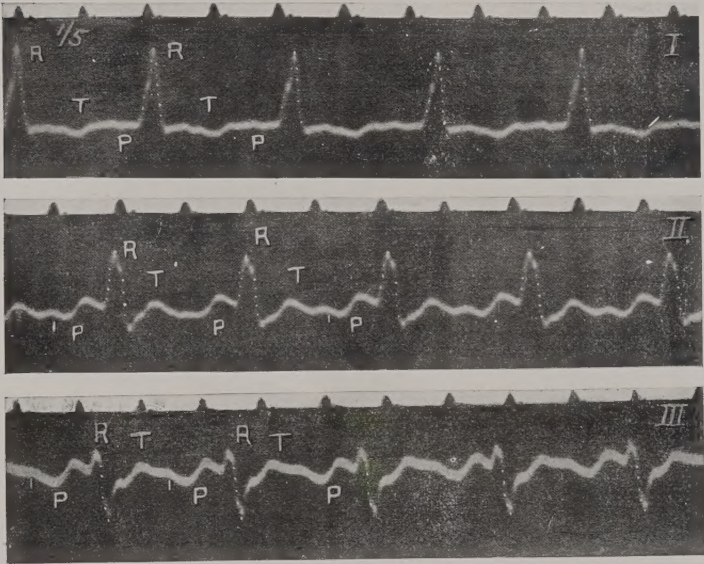


FIG. 9.

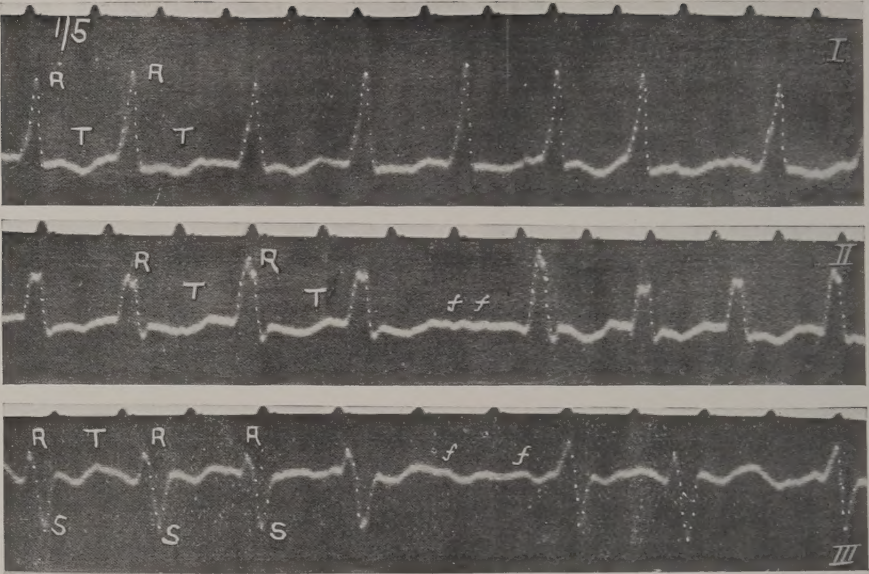


FIG. 10

Fig. 11-14. *CASE 2.* A series of curves taken on July the 4th, 1911. The time-marker is in 1/30 sec..

Fig. 11 shows four beats of the normal rhythm, each cycle consisting of *P*, *R* and *T* variations. The amplitude of *R* is variable.

Fig. 12. A curve taken from a slow period and showing a single premature contraction, arising from an ectopic auricular focus. The premature auricular representative is marked *P* below the curve.

Fig. 13. A curve taken during an auricular paroxysm showing inverted *P* waves. The last cycle but one is constituted by a beat arising from a second focus, as is shown by the shape of the auricular representative marked *P'*. Note the variation in the amplitude of *R* in this figure.

Fig. 14. The end of a regular paroxysm of the same type. Two beats of the usual paroxysmal form are shown. The third beat ends the paroxysm and arises, as shown by the shape of *P'*, from a second abnormal focus. The post-paroxysmal pause and the resumption of the normal rhythm are seen.

Fig. 15. *CASE 2.* Taken on June the 27th, 1911, and showing a single premature auricular contraction which springs from the same focus as the majority of the paroxysmal beats. The time-marker is in 1/5 sec..

Fig. 16-18. *CASE 2.* A series of curves taken on July the 18th, 1911. Fig. 7 covers the same period of observation. The time-marker in all three curves is in 1/30 sec..

Fig. 16 was taken shortly after the onset of a regular paroxysm. The type of paroxysm is similar to that shown in Fig. 13.

Fig. 17 was taken almost directly afterwards. It shows no evidence of a co-ordinate auricular contraction; the heart-beat is quite irregular and oscillations characteristic of auricular fibrillation are present (*f, f*).

Fig. 18. A similar curve taken later during the same attack, and showing the characteristic picture of auricular fibrillation.

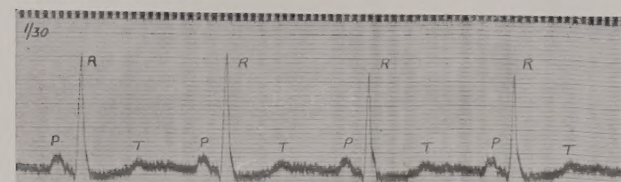


FIG. 11

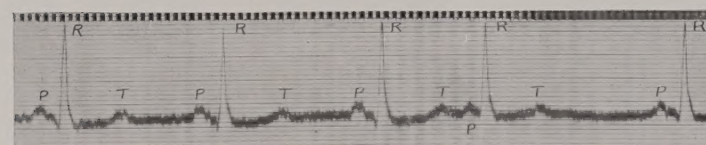


FIG. 12.

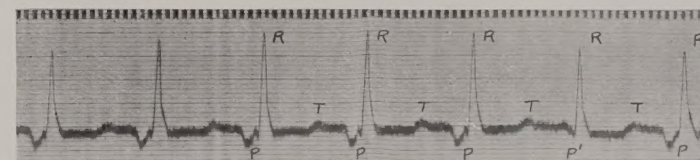


FIG. 13.

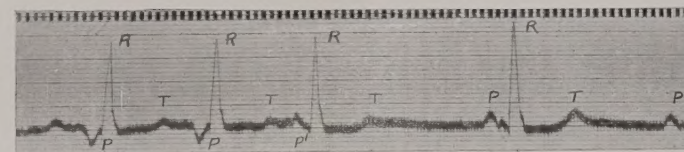


FIG. 14.

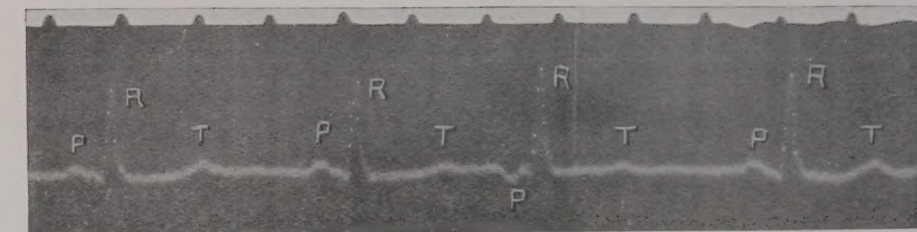


FIG. 15.

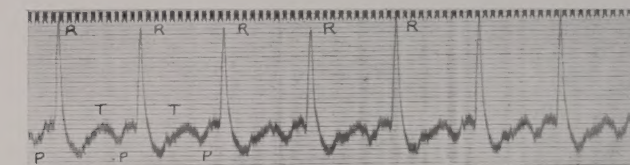


FIG. 16.

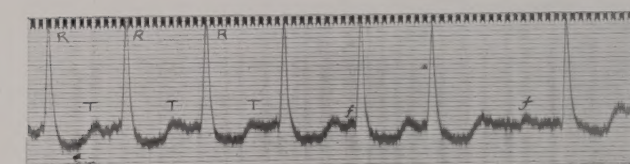


FIG. 17.

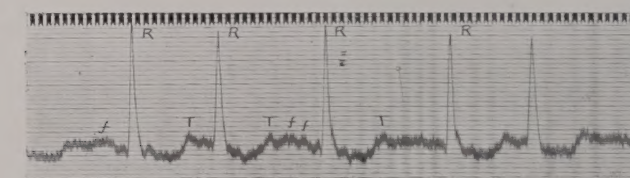


FIG. 18.

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